Evaluation of Beet Leafhopper Transmitted Virescence Agent Damage in the Columbia Basin

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Abstract Potato purple top disease is caused by a phytoplasma known as Beet Leafhopper Transmitted Virescence Agent (BLTVA), which is vectored by the beet leafhopper (BLH, *Circulifer tenellus* Baker). Previous studies determined that BLTVA can cause significant reductions in yield and tuber quality; however, quantifying the damage caused by BLTVA and the insect vector has been challenging. In 2009–2011, potato plants at different growth stages were exposed to varying densities of BLH in a screen house located at the Hermiston Agricultural Research and Extension Center in Hermiston, OR. The densities of potentially infective BLH were one BLH per plant (low), two BLH per plant (medium), and five BLH per plant (high). Releases occurred at the following growth stages: vegetative, tuber initiation, tuber bulking, and maturation. The treatments were arranged in a randomized complete block design with three replications per treatment. Disease incidence was monitored weekly and yield was assessed. When all 3 years were combined, we found that increasing rates of disease incidence correlated with decreasing yields. We also found that greater yield losses were observed with later BLH release times. With both correlations, differences between years were a strong contributing factor.

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There was a mean decrease in yield of 0–12 % at a density of one BLH per plant, 6–19 % at two BLH per plant, and 6–20 % for five BLH per plant. These general trends in yield loss suggest that economically relevant damage may occur at levels as low as one or two potentially infective BLH per plant in the Columbia Basin.

Resumen La enfermedad de la punta morada es causada por el fitoplasma conocido como agente de virescencia transmitido por la chicharrita de la remolacha (BLTVA por sus siglas en inglés). El vector es el chicharrita saltador conocido como chicharrita de la remolacha (BLH, *Circulifer tenellus* Baker). Estudios previos determinaron que BLTVA reduce la calidad del tubérculo y la producción del cultivo de la papa; sin embargo, la determinación de valores de daño han sido difíciles de investigar. Para responder esta pregunta, en la temporada de campo del 2009–2011, plantas de papa de diferentes estados de desarrollo fueron expuestas a diferentes densidades de la plaga en un invernadero en la Estación Experimental de Investigación y Extensión de Hermiston (Hermiston Agricultural Research and Extension Center) en Hermiston, OR. Los niveles de infestación fueron de un BLH por planta (nivel bajo de infestación), dos BLH por planta (nivel medio de infestación), y cinco BLH por planta (nivel alto de infestación). Las infestaciones ocurrieron en la etapa vegetativa del cultivo, a la iniciación de la tuberización, durante la formación del tubérculo y durante la maduración del tubérculo. Los tratamientos fueron organizados como bloques al azar con tres repeticiones por tratamiento. La incidencia de la enfermedad fue monitoreada cada semana y la producción fue estimada. Datos combinados sugieren que el incremento en el número de BLH por planta correlaciona positivamente con la producción del cultivo. La diferencia entre años también influyó para ambas correlaciones. La disminución en la producción del cultivo correlaciona con infestaciones tardías de BLH. Hubo una diminución en la producción del cultivo de 0–12 % con infestaciones de un BLH por planta, de 6–19 % con dos BLH por planta y de 6–

20 % con cinco BLH por planta. Datos sugieren que el daño económico occurre a un nivel de uno o dos BLH por planta en la zona de la rivera del Columbia.

Introduction

The Columbia Basin of Oregon and Washington produces the highest potato yields worldwide mainly due to long, hot summer days and cool nights (Strand et al. 2006). Potatoes in Oregon and Washington are valued at about $12,400 per hectare (NASS 2011). Field size under the center-pivot irrigation, the most common irrigation system used in the Columbia Basin, can vary from 8 to over 80 ha, which could represent a value of more than $1,000,000 per field (Bohl and Johnson 2010). Insects such as the beet leafhopper (BLH), *Circulifer tenellus* Baker (Hemiptera: Cicadellidae) are almost a unique pest of the Columbia Basin agro-ecosystem; though they are also a pest in areas of California (Severin 1930). Beet leafhoppers periodically cause serious economic damage to potatoes grown in the region (Hamm et al. 2003; Munyaneza et al. 2005). This insect transmits a phytoplasma known as Beet Leafhopper Transmitted Virescence Agent (BLTVA), or 'Columbia Basin purple top phytoplasma,' which causes purple top disease (Crosslin et al. 2005; Golino et al. 1987; Munyaneza et al. 2006; Munyaneza et al. 2007). Purple top disease is characterized by shortened internodes, curling and purpling of the foliage, aerial tubers, as well as reduced yields (Golino et al. 1987; Munyaneza et al. 2007). The foliar symptoms associated with BLTVA resemble several other diseases, including zebra chip, psyllid yellows, aster yellows, or potato leafroll virus (Burkness et al. 1999; Gabelman et al. 1994; Hamm et al. 2003; Munyaneza et al. 2008b). Many of these diseases, including BLTVA, can be identified using polymerase chain reaction (PCR) analyses (Crosslin et al. 2006; Crosslin and Hamlin 2011).

Beet leafhoppers feed on weed hosts through the fall, winter and spring (Cook 1942; Hills 1937). The preferred hosts for BLH are not potatoes, but weed species, including redstem filaree (*Erodium cicutarium* L.), Russian thistle (*Salsola iberica* Sennen), tumble mustard (*Sisymbrium altissimum* L.) and flixweed (*Descurainia sophia* L.) (Cook 1967; Hills 1937; Severin 1930). During the hot summer months these weed hosts naturally senesce, forcing BLH into irrigated crops, mainly potatoes (Cook 1942; Murphy et al. 2012). Beet leafhoppers do not reproduce well on potatoes (Cook 1967; Hills 1937), though populations in the Columbia Basin persist through the growing season and into the fall and winter (Crosslin et al. 2012; Munyaneza et al. 2008a; Murphy et al. 2012). Beet leafhopper populations are monitored throughout the season using yellow sticky cards, but economic thresholds for beet leafhopper populations have not been established (Crosslin et al. 2012). Another concern is whether growers even need to spray to control BLH and BLTVA. Currently, growers rely on monitoring with imprecise thresholds or calendar spray regimes from May through mid to late June (Schreiber 2003; Schreiber et al. 2012). More accurate assessment of the damage caused by BLH and BLTVA should provide a better estimate of when insect numbers warrant control and aid implementation of integrated pest management programs. Thus, the main objective of the present study was to provide a more accurate assessment of the impacts of BLTVA on yield in the Columbia Basin.

Methods

Experimental Design

This 3-year study (2009–2011) was conducted in a screen house (i.e., a greenhouse with 1 mm²-screening instead of glass) at the Oregon State University Hermiston Agricultural Research and Extension Center (OSU-HAREC) in Hermiston, Oregon. The structure was built to surround existing ground and the soil is Adkins fine sandy loam. Prior to establishment of the screen house and conversion to potato production, the ground was maintained as turf. Certified seed potatoes (cv. Umatilla) were hand-planted in small plots consisting of 1.8 m of a single row each. Six seed potatoes were planted in each plot. The plots were arranged in a randomized complete block design and each treatment was replicated three times. Plots were also covered individually with a 2.4 m tent-like narrow cage designed to cover a single row of potato plants. Each cage was made of a frame of 4.6-m fiberglass tree stakes (GEOTEK, Inc., Stewartville, MN) covered with 1 mm× 0.5 mm fine insect screen (USGR, Inc., Seattle, WA). The plots were covered before potato emergence and the bottoms of the cages were buried in the ground to exclude unwanted insects. Irrigation was accomplished with sprinklers and fertilizer was applied following standard agronomic practices for the Columbia Basin. A pre-plant herbicide was used to control weeds, and selective insecticide/miticides and herbicides were applied as needed (Table 1); while the insect screen excluded most insects, it did not exclude thrips or spider mites.

Insect Releases and Density Studies

Each year BLH were released into the cages described above at low (one BLH/plant), medium (two BLH/plant) or high (five or more BLH/plant) densities. Insects were permitted to remain on the plants indefinitely after release. Control plots did not receive BLH and were caged to exclude insects. Since experimental conditions were designed to closely simulate the

Table 1 Agronomic practices in the screen house experiment 2009–2011, Hermiston, OR

Management	2009	2010	2011
Vapam		4 November 2009	3 November 2010
Pre-plant fertilizer		14 April: 25 lb	18 May: 25 lb
Planting date		17 May	5 June
Pre-plant herbicide	Glyphosate	18 May: Matrix and Dual	23 May: Matrix and Dual
Setting cages	15 May	12 May	5 June
Beet leafhopper releases			
1st release	24 June	3 June	7 July
2nd release	10 July	23 June	21 July
3rd release	28 July	30 June	4 Aug
4th release	NA	14 July	NA
Irrigation			
After crop emergence	Mon., Wed, Fri. and Sun. 45 min	Every other day - 20 min	24 June: 40 min Mon., Wed., Fri.
	Starting 20 Aug, 60 % water	Starting 22 July, 30 min every day	6 July: 20 min Mon., Wed., Fri.
Fertilizer			
Starting:	16 July	14 June	1 July
30 lb N	3 Aug	18 June	8 July
30 lb N	6 Aug	25 June	15 July
30 lb N	10 Aug	2 July	22 July
30 lb N	17 Aug	9 July	29 July
30 lb N	24 Aug	16 July	5 August
30 lb N	31 Aug	23 July	12 August
30 lb N	7 Sept	30 July	19 August
30 lb N			26 August
Insecticides			
Success		None	10 August
Miticides			
		24 July: Oberon	6 July: Acramite
			12 August: Acramite
Herbicides			
Glyphosate	Before planting	4 August	6 July
Fusilade	14 July		
Matrix and Sencor	21 July		

situation in the field, BLH were collected directly from field populations in 2009 and 2010. Phytoplasma in field populations of BLH ranges from 5 to 34 % in the Columbia Basin (Munyaneza et al. 2010a). In 2011, natural BLH populations, and the incidence of BLTVA were low. In order to secure BLH populations for releases, we used BLH from a colony where insects were "forced" to feed on BLTVA-infected plants by placing them in small cages fastened around symptomatic portions of a plant. Colonies were reared on preferred hosts, including beets and radishes, as well as periwinkle plants, which can maintain a high titer of BLTVA (Golino et al. 1989). Insects in the colony were maintained at approximately 25±7 °C with 14:10 D:L. Prior to release, a sub-sample of BLHs ($n = 14$) were collected from the colony and tested individually for BLTVA as described by Crosslin et al. (2006). The mean percent of infected BLH from the colony in 2011 was 23.3 %, prior to release. These rates of infection simulated natural populations in the field.

Insect Releases and Release Timing

Releases occurred at different growth stages of the plant: 1) vegetative (II), 2) tuber initiation (III), 3) tuber bulking (IV) and 4) maturation (V). There were three, four, and three releases in 2009, 2010, and 2011, respectively. The final releases in 2009 and 2011 were omitted due to alternative weed hosts in the cages and imminent harvest of the plants, respectively.

Table 2 Mean percent yield loss per plot for different BLH densities. Treatments were compared to the controls (BLH-free plots) in each replication to calculate yield loss; values are the difference between the mean yield for each treatment and the control. Release densities are low (one BLH/plant), medium (two BLH/plant), and high (five BLH/plant)

Density	Low	Medium	High
2009	10.75±29.6 %	−5.59±27.2 %	−5.73±15.5 %
2010	−12.38±11.3 %	−18.86±7.0 %	−19.86±10.5 %
2011	−6.76±3.8 %	−8.00±3.6 %	−10.79±2.7 %

Disease Incidence

Disease incidence (foliar symptoms) was assessed visually by the same person on a weekly basis in 2009, 2010 and 2011. In 2010 and 2011, no foliar symptoms were apparent. Thus, in 2010 a sub-sample of the plants were tested to detect BLTVA using PCR as described by Crosslin et al. (2006). In 2011, all plants were sampled weekly and potentially symptomatic plant tissue was targeted during sampling. Although samples were collected weekly, PCR analysis was only performed every 2 weeks, as previously infected plants were expected to test positive again. Control plots never exhibited visual or molecular evidence of infection with BLTVA.

Yield Data

Potatoes were dug by hand and sorted by weight classes (i.e., < 0.057 kg, 0.057–0.113 kg, 0.113–0.227 kg, 0.227–0.340 kg and >0.340 kg) to estimate yield per plot (kg). The specific gravity was measured for each plot for all 3 years by weighing the tubers in air and then again in water. Specific gravity is used as a measure of dry matter content and positively correlates with yields of finished product (i.e., chips, fries, etc.) (Lulai and Orr 1979).

Data Analysis

The data for the screen house experiments were analyzed separately by year and combined. Disease incidence was analyzed using a mixed, binomial general model ANOVA and chi-

squared analysis (PROC GENMOD). Models were adjusted by including or excluding additional factors to minimize type 1 error due to overdispersion. For example, the factors release timing and BLH density for 2010 and 2011 were analyzed separately to achieve the most robust model and correct for overdispersion (SAS 2012). Significant models were further analyzed using contrasts, followed by a Holm-Sidak adjustment (Abdi 2010; Holm 1979; Šidák 1967). The yield data were analyzed using a mixed model ANOVA (PROC MIXED) with combination treatments: BLH density × release timing. The untreated plots for the fourth release in 2009 and 2011 were not pooled with the control plots, but maintained as a separate treatment for these analyses. These analyses were performed using SAS 9.2. The combined data for all 3 years were also analyzed using a regression analysis to identify correlations between mean disease incidence, BLH release timing, BLH densities and yield. Since the different methods measured the same parameters (i.e., disease incidence), the combined data were deemed appropriate for analyzing correlations. Disease incidence was combined so that a positive result for either measurement was counted as evidence of the disease. For correlations between release timing and yield, release timing was converted to cumulative degree days, and the controls (i.e., no degree day value for release) were excluded. Following regression analyses, mean yield, mean disease incidence and transformed mean growing degree days were analyzed for differences between years with and without the controls, to match each regression analysis, using one-way ANOVA. The percent yield loss, compared to the control plots, was also analyzed for differences between BLH density and release timing using ANOVA. Data were transformed when they failed to meet normality assumptions for ANOVA or regression analyses. The growing degree day data were transformed using a Johnson transformation. These analyses were done using Minitab 16.

Results

There were no significant differences in mean yield loss based on BLH release densities: low, medium and high ($F=0.26$;

Table 3 Mean percent yield loss per plot for different BLH release timings. Treatments were compared to the controls (BLH-free plots) in each replication to calculate yield loss; values are the difference between

the mean yield for each treatment and the control. Releases occurred at the following growth stages: 1) vegetative, 2) tuber initiation, 3) tuber bulking, and 4) maturation

Release	Potato growth stage			
	1 Vegetative	2 Tuber initiation	3 Tuber bulking	4 Maturation
2009	5.64±30.7 %	3.55±14.7 %	−9.76±26.5 %	–
2010	−10.29±15.4 %	−19.66±7.6 %	−13.33±9.6 %	−24.86 %±11.4
2011	−8.30±3.5 %	−5.89±3.3 %	−11.36±3.3 %	–

Table 4 Mean BLTVA disease incidence as a proportion per plot in the screen house experiments for 2009, 2010, and 2011. Data was combined for visual and PCR detection techniques. Releases occurred at the following growth stages: 1) vegetative, 2) tuber initiation, 3) tuber bulking, and 4) maturation

Treatment	2009	2010	2011
Release 1—Low	0.89±0.06	0.0±0.00	0.22±0.15
Release 1—Med	0.56±0.06	0.0±0.00	0.0±0.00
Release 1—Hi	0.72±0.15	0.0±0.00	0.0±0.00
Release 2—Low	0.78±0.15	0.0±0.00	0.06±0.06
Release 2—Med	0.61±0.31	0.0±0.00	0.24±0.12
Release 2—Hi	0.67±0.25	0.0±0.00	0.11±0.06
Release 3—Low	0.28±0.15	0.0±0.00	0.0±0.00
Release 3—Med	0.39±0.20	0.0±0.00	0.0±0.00
Release 3—Hi	0.61±0.06	0.06±0.06	0.0±0.00
Release 4—Low	–	0.0±0.00	–
Release 4—Med	–	0.11±0.11	–
Release 4—Hi	–	0.0±0.00	–

df=3, 89; P=0.856). The mean yield loss for each plot, based on the density of BLH, is shown in Table 2.

There were no significant differences in mean yield loss due to BLH release timing during plant growth stages: vegetative, tuber initiation, tuber bulking or maturation (F=0.68;

df=4, 88; P=0.606). The mean yield losses based on release timing are shown in Table 3.
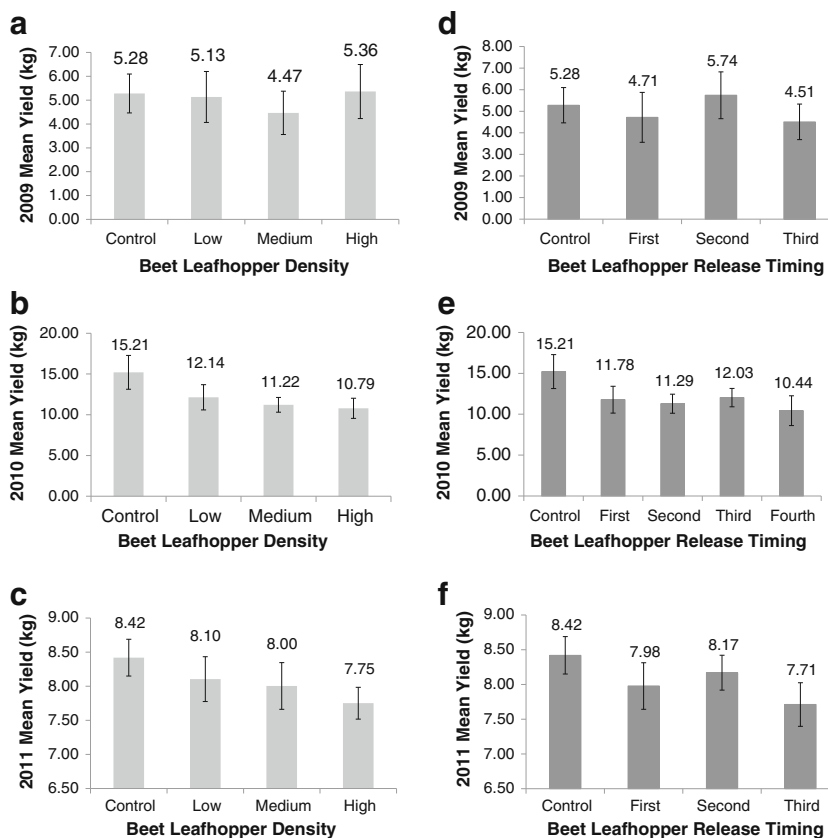
Disease Incidence

Disease incidence data is shown in Table 4. In 2009 there were no significant differences between treatments in disease incidence (foliar symptoms), either based on release timing (F= 2.85; df=3, 22; P=0.061) or BLH densities (F=0.69; df=2, 22; P=0.511). In 2010 and 2011, no apparent foliar symptoms were observed and disease incidence was measured using PCR.

In 2010 disease incidence was significantly different for release timing (F= 2.95; df=3, 30; P=0.048), but not BLH density (F=1.98; df=2, 31; P=0.155). When release timings were compared using contrasts, there were no significant differences after the Holm-Sidak adjustment.

In 2011 disease incidence was significantly different for release timing (F=5.51; df=2, 25; P=0.010), but not for BLH density (F=0.64; df=2, 32; P=0.536). When release timings were compared using contrasts, plants in the second release (tuber initiation) exhibited significantly higher disease incidence than the third release (tuber bulking) (F=10.95; df=1, 25; P= 0.003). Plants in the first release (vegetative stage) also exhibited significantly greater disease incidence compared to plants in the third release (tuber bulking) (F=6.14; df=1, 25; P=0.020).

Fig 1 Mean yield (kg) by BLH density (**a**, **b**, **c**) or release (**d**, **e**, **f**) and year. Years are **a** and **d**, 2009; **b** and **e**, 2010; **c** and **f**, 2011. Controls are BLH-free plots
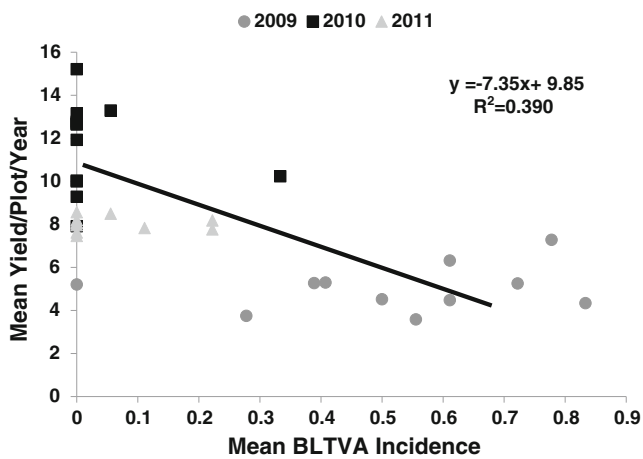
Fig. 2 Correlation between potato yields and BLTVA disease incidence (visual and PCR combined) in 2009, 2010, and 2011 combined

Yield

There were no significant differences in yield between treatments (BLH densities × release timing) in 2009 ($F=0.46$; df=10, 27; $P=0.900$), 2010 ($F=0.73$; df=12, 25; $P=0.714$), or 2011 ($F=0.52$; df=10, 27; $P=0.864$) (Fig. 1).

When the data for all 3 years were combined, there was a significant correlation between disease incidence and yield ($F=22.70$; df=1, 33; $P<0.001$). Higher yields were associated with lower disease incidence. Differences between years contributed to the relationship (Fig. 2), as there were significant differences between years for mean yield ($F=68.64$; df=2, 32; $P<0.001$) and mean disease incidence ($F=35.68$; df=2, 32; $P<0.001$) when controls were included. Yield also correlated with release timing, measured in cumulative growing degree days ($F=20.13$; df=1, 28; $P<0.001$). Plants exposed to BLH at earlier dates had higher yields. Differences between years contributed to the relationship (Fig. 3), as there were significant differences between years for mean yield ($F=60.12$; df=2, 27;
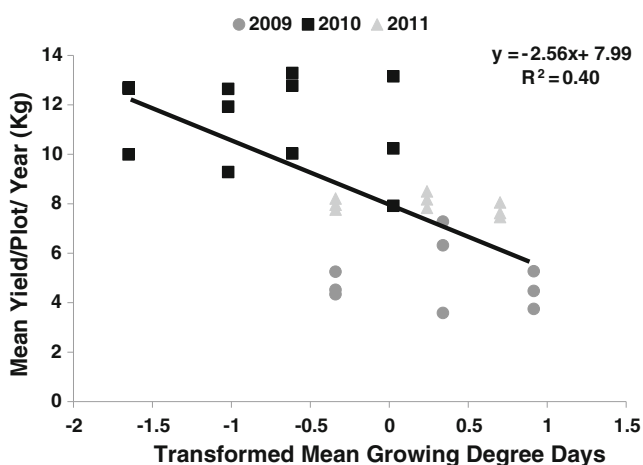

Fig. 3 Correlation between potato yields and BLH release timing in 2009, 2010, and 2011 combined

$P<0.001$) and transformed mean growing degree days ($F=13.15$; df=2, 27; $P<0.001$) when controls were excluded. There were no significant differences in percent yield loss for BLH density or release timing.

Discussion

This research demonstrates that BLH populations infected with BLTVA can reduce potato yields in the Columbia Basin. A low density of BLH (one BLH/plant) at the infectivity rate of approximately natural populations can result in an average of 3 % yield loss. Medium BLH pest-pressure (two BLH/plant) and high BLH pressure (five BLH/plant) can result in an average of 12 % yield loss. In an 80-ha field, 12 % yield loss translates as a loss of $120,000 (Bohl and Johnson 2010; NASS 2011). These results, though not statistically significant, suggest that economically relevant damage may occur at the level of only one or two potentially infective BLH per plant in the Columbia Basin. Currently, growers rely on calendar sprays or anecdotal thresholds of 40–100 BLH/sticky card/week for beet leafhopper control in potatoes (Schreiber et al. 2012). Our data provides growers with more information regarding BLH and BLTVA damage than was previously available.

It has been suspected that infection with BLTVA at earlier growth stages would cause greater decreases in yield (Munyaneza et al. 2010b; Schreiber et al. 2012). Munyaneza et al. (2010b) established that younger plants experienced higher infection rates in laboratory and field studies. Though yields were not measured in the study by Munyaneza et al. (2010b), it was logically theorized that higher rates of infection would result in greater yield reductions. While the plants in this study showed similar trends in susceptibility (i.e., more plants were infected during early releases on younger plants as compared to later releases on older plants), the results of this study do not lend support to or directly contradict the previous hypothesis regarding yields. According to the results of the current study, it seems that infection later in the season could have a greater impact on yield. However, the relationship between yield and infection-timing may be an artifact of differences between years, particularly in the yield. The yield in this small-plot study could have been influenced by many different factors each year including temperatures, water, nitrogen, soil compaction, plant infection rates and BLH infection rates. As a result of the differences evident between years in the current study, it is impossible to lend further support to the current hypotheses regarding yields and infection timing. Future research is necessary to clarify the relationship between potato yields and the timing of BLTVA infection.

Infected plants from early in the season in 2011, where plants were sampled for BLTVA weekly, did not consistently test positive for the disease later in the season. While we are uncertain of the exact cause, there are several possible explanations

for these observations that should be discussed. First, we thought it might be possible that positive samples were actually false-positives. However, as multiple samples were tested a second time and tested positive again, false-positives are an unlikely explanation. Alternatively, there is extreme variability, up to five orders of magnitude, in the amount of phytoplasma in an individual plant, even one that shows symptoms (Crosslin et al. 2006). Crosslin et al. (2006) measured the sensitivity of the nested PCR procedure used in this study, and it was less sensitive to these fluctuations in BLTVA titers compared to a real-time PCR procedure, which may account for the samples that failed to test positive later in the season (i.e., phytoplasma titers remained low as the plant increased in size). Another possible contributor is the environment. According to other research on phytoplasmas, infection establishment and detection can be hampered by low temperatures or dual infections (De Oliviera et al. 2007; Golino et al. 1987). Temperatures during the summer of 2011 were extremely mild compared to previous years: maximum air temperatures did not exceed 38 °C, as is typical (Agrimet 2012). It is possible that these uncharacteristically low temperatures inhibited disease development, making detection using a nested PCR less effective. Another explanation is related to the development of symptoms and the disease in the host plant. It is not uncommon for infected plants to be asymptomatic (Lee et al. 2004). BLTVA disease and symptom development varies considerably between hosts and can take as long as 14 weeks (Golino et al. 1987; Golino et al. 1989). Plants with minimal or no symptoms can still harbor BLTVA that may be acquired by BLH (Golino et al. 1989). Collecting tissue samples 1 week after infestation may not have allowed sufficient time for infection, multiplication, and translocation of the phytoplasma. It is possible that in sampling the plant, specifically any potentially symptomatic tissue, all the infected plant material was removed, leaving no source of infection for the next sampling. Inevitably, as plant size increases, it becomes less likely that localized infections will be detected or sampled. It may be that a combination of these different possibilities, or even other unknown factors, contributed to the observed results. Future research on BLTVA disease development and dynamics is warranted in the Columbia Basin. Prevalence of the disease is extremely variable and unpredictable between seasons, and it is most likely influenced by multiple biotic and abiotic factors (Munyaneza et al. 2010a).

Future research is also necessary to further refine and incorporate the damage estimates reported in this study so that they may be used with common monitoring techniques like D-VAC sampling (inverted leaf blower) or standard yellow sticky cards (Munyaneza et al. 2008a; Murphy et al. 2012). As BLH populations are highly mobile and variable, more research is also required regarding their migration within the Columbia Basin. We are uncertain about whether outbreak populations in a particular field originate on nearby weeds, or in remote desert scrubland. Populations in California have been documented to undergo long-distance migrations (Cook 1967), but no similar research has been done in the Columbia Basin. As BLH populations fluctuate considerably between years (Crosslin et al. 2012; Munyaneza et al. 2008a; Murphy et al. 2012), it will be interesting to monitor their impact on potato production in the Columbia Basin in the future.

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